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Trunk Muscle Recruitment Patterns in Patients With Low Back Pain Enhance the Stability of the Lumbar Spine

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Study Design. A comparative study of trunk muscle recruitment patterns in healthy control subjects and patients with chronic low back pain was conducted.

Objective. To assess trunk muscle recruitment in patients with low back pain.

Summary of Background Data. Conflicting evidence has been reported on the level and pattern of trunk muscle recruitment in patients with low back pain. The disparities can be explained partly by methodologic differences. It was hypothesized that trunk muscle recruitment patterns may be altered in patients with low back pain to compensate for reduced spinal stability.

Methods. For this study, 16 patients with low back pain and 16 matched control subjects performed slow trunk motions about the neutral posture and isometric ramp contractions while seated upright. Ratios of electromyographic amplitudes and estimated moment contributions of antagonist over agonist muscles and of segmentally inserting muscles over muscles inserting on the thorax and pelvis only were calculated. In addition, model simulations were performed to assess the effect of changes in muscle recruitment on spinal stability.

Results. The ratios of antagonist over agonist, and of lumbar over thoracic erector spinae electromyographic amplitude and estimated moment contributions were greater in the patients than in the control subjects. The simulation model predicted that these changes would effectively increase spinal stability.

Conclusions. Trunk muscle recruitment patterns in patients with low back pain are different from those in healthy control subjects. The differences are likely to be functional with respect to enhancement of spinal stability in the patients. [Key words: electromyography, low back pain, spinal stability, trunk muscle recruitment] **Spine 2003;28:834–841**

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Trunk muscle recruitment patterns in patients with low back pain (LBP) have been studied in a wide range of tasks and patient groups.[1,2] Most of the early studies were motivated by the pain–spasm–pain model first proposed by Travel *et al.*[3] This model assumes that pain reflexively induces sustained muscle activation, which in turn causes pain, thus establishing a vicious circle. Although some studies appear to support this model by showing higher trunk muscle electromyographic (EMG) amplitudes in patients with LBP than in healthy control subjects,[2] other studies show ambiguous results or even reduced EMG amplitudes.[1]

Some of these disparities can be explained by methodologic problems, an important one of which is the normalization of EMG data. In general, absolute EMG amplitudes depend on many factors unrelated to the level of muscle activation, such as thickness of tissues overlying the muscle and skin impedance. To obtain a signal independent of such factors, normalization of EMG amplitudes to the amplitudes obtained in maximum voluntary contractions (MVC) often is used. However, this procedure is considered unreliable for patients because patients usually are unwilling or unable to perform maximum contractions. Normalization to submaximal contractions does not provide a solution because it can be expected that in patients, the EMG amplitudes during these submaximal tests will be affected similarly to the levels during the activities to be studied.

Another explanation for the disparate results stems from the differences in motor tasks during which the EMG measurements were made. Lund *et al*[1] concluded that there is evidence for increased activation during tasks in which the muscle studied acts as an antagonist, whereas usually no increased activation is found in static postures, and even decreased activation is found when the muscle acts as agonist. These findings led these authors to propose the pain adaptation model. In short, nociceptive afferents are thought to exert both excitatory and inhibitory influences on the alfa motor neuron pool through interneurons. The influence of these interneurons is modulated by the central command controlling a movement, which explains the switching from inhibition to excitation when muscles change roles from agonist to antagonist. The changes in agonist and antagonist activation lead to reduced movement speed and range of motion. Induced pain experiments have lent considerable credence to the pain adaptation model.[4–7] However, some experimental data suggest that with respect to low back pain, refinement of the model is needed.

First, the relation between pain state and muscle activation is not as direct as suggested by the theory. Arena *et al*[8] did find differences in trunk extensor activation between patients and controls, but found no relation between pain state and level of activation in the patient group. Svensson *et al*[9] found induced pain to outlast the changes in muscle activation. Second, some of the differences between patients with LBP and control subjects are not well captured by the prediction that muscle activation is increased only when the muscle functions as antagonist. Some studies show increased activity in static postures, including upright stance[8,10,11] and standing with full trunk flexion.[11–15] During the swing phase in walking, when trunk muscles normally are silent, both left and right trunk extensor muscles show increased activation in patients.[4] In these cases, the muscles studied act to support posture against gravity instead of acting as antagonist. Furthermore, the fact that increased activation is found in static postures implies, in view of static equilibrium requirements, that cocontraction of the muscle on opposite sides of the joints occurs.

The current authors surmise that the changes in muscle activity described should be regarded as functional adaptations to a reduced spinal stability in patients with LBP. Panjabi[16] first suggested that instability of the spine likely results from any dysfunction of either spinal structures or trunk muscles or from reduced neural control over the latter, and as such, is an important aspect of LBP. Instability of the spine could lead to excessive tissue strain and consequent pain. Panjabi[17] further hypothesized that muscle activity could be used to compensate for a loss of passive stability. It has been shown, both by modeling and experimentation, that muscles can contribute to stability of the trunk through cocontraction.[18–22] In addition, healthy subjects increase cocontraction in response to conditions that threaten spinal stability.[23,24] It is likely that such adaptation could be triggered by information from both mechanoreceptors[25] and nociceptors. This would explain why the relation to pain is not straightforward.

The current study focused on the patterns of trunk muscle recruitment in standardized tasks rather than the level of muscle activation. Patterns of trunk muscle activation can be described by EMG amplitude ratios of different muscles,[26] thus circumventing the normalization problem referred to earlier.

On the basis of the assumption that patients with LBP adapt muscle recruitment to compensate for a loss of stability, the authors have formulated several hypotheses on the pattern of activation and on force sharing between these muscles. They expected the patients to show more cocontraction than the control subjects. Therefore, the first hypothesis is that the ratios of antagonist over agonist muscle activation are higher in the patients. Furthermore, Bergmark[27] and Crisco and Panjabi[28] used mechanical modeling to show that activation of segmentally inserting muscles would more effectively subserve spinal stability than activation of muscles inserting on the thorax and pelvis only. Following this suggestion, the current authors hypothesized that their study patients would show higher activation of the lumbar erector spinae relative to the thoracic erector spinae. In addition, they hypothesized that the ratios of internal oblique muscle activation and moment contribution over rectus abdominus muscle activation would be increased in the patients.

Any increase in moment contribution of one synergist relative to another will be reflected in a change in the ratio of their EMG amplitudes. In contrast, an increase in antagonistic moment production is not necessarily reflected in an increase of the antagonist over agonist EMG ratio. This depends on the ratio of moment-producing capacity of the two muscles per unit of EMG amplitude.[29] Therefore, in addition to the use of EMG ratios, an attempt was made to estimate the mechanical contribution of several trunk muscles to the total moment by using an EMG-assisted model. Conventionally, MVC-normalized EMG amplitudes are fed into these models as an estimate of muscle activation. In view of the normalization problem referred to earlier, the authors developed a model that uses a series of contractions in which known moments are produced to obtain normalization not based on maximum contractions.

Finally, to verify whether the aforementioned changes in relative contributions to the net moment produced do indeed contribute to spinal stability, model simulations were performed to calculate the index of spinal stability, as proposed by Cholewicki and McGill[30] for both a generic healthy subject and a patient.

■ Methods

Participants. For this study, 16 patients with chronic idiopathic LBP and 16 matched healthy control subjects volunteered and signed the consent form approved by the Yale University Human Investigation Committee. Low back pain was defined as a persisting or periodic pain with a duration longer than 6 months. The patients with LBP included in this study had no neurologic deficits, structural deformities, genetic spinal disorders, or previous spinal surgery. Their radiographs showed only normal, age-related changes. These patients had experienced LBP for periods ranging from 6 months to 35 years. Their pain intensity had varied from mild to severe, with some pain-free intervals. On a 10-cm visual analog scale, the patients expressed their LBP on the day of testing as 2.1 ± 1.2. The consumption of analgesics, mostly nonsteroidal antiinflammatories, varied from daily use to medication as needed. The Roland Disability Questionnaire, on the average, showed low scores (5.6 ± 4.4 of 24), reflecting the ability of all the patients to continue working, with some sick leave taken only for days of intolerable pain. All the patients were screened by an orthopedic surgeon before the testing to ensure that the inclusion criteria were met.

Healthy control subjects, recruited *via* advertisement, matched the experimental LBP group by gender, age, weight, and height (Table 1). These control subjects had never experienced back pain with a duration longer than 3 consecutive days.

Table 1. Anthropometric Data of Subjects

	Males		Females	
	Controls	LBP Patients	Controls	LBP Patients
N	11	11	5	5
Age (yrs)	36 (13)	35 (13)	45 (10)	44 (7)
Weight (kg)	77.5 (19.1)	82.2 (12.9)	58.8 (13.1)	68.2 (12.3)
Height (cm)	176 (9)	182 (7)	168 (8)	162 (9)
T9–L4 dist (cm)	19.7 (3.1)	21.4 (3.5)	21.4 (1.5)	19.6 (4.4)

Standard deviations are given between parentheses. There were no significant differences between patients with LBP and healthy control subjects ($P > 0.05$).

Procedure. The study participants were placed in a semiseated position in an apparatus that restricted hip motion but left their upper torso free to move in any direction. They were asked to perform slow sagittal, frontal, and transversal plane movements in both directions around a neutral spine posture from approximately a −20° to a 20° angle. Trunk motion took place from one end position, continued slowly through neutral posture, and ended in the opposite position. The participants completed one or two practice trials to learn to pace their trunk motion over the 7-second trials. The trials were repeated with 16 kg of mass added to the torso for the men and 8 kg added for the women. For that purpose, a chest harness with two pouches in front and two pouches on the back of the chest, placed at approximately T9, was filled with lead shot.

In addition, participants performed isometric ramp contractions in left and right lateral bending, flexion, and extension up to a level that they experienced as requiring effort but tolerable. A steel cable attached to a chest harness at approximately T9 and to a load cell provided the resistance. All the motion trials and ramp contractions were performed twice.

Instrumentation. Trunk angle was measured with an electromagnetic sensor device (Flock of Birds, Ascension Technologies Corp., Burlington, VT, USA) at 80 Hz. The sensor was placed at approximately T9 on the back.

The 12 EMG signals were collected using disposable pellet Ag-AgCl surface electrodes (diameter, 1 cm; interelectrode distance, 3 cm). After the skin was abraded and cleaned with alcohol, the electrodes were placed over the following trunk muscles bilaterally: rectus abdominus (3 cm laterally to the umbilicus), external oblique (approximately 15 cm laterally to the umbilicus), internal oblique (approximately midway between the anterior superior iliac spine and the symphysis pubis, above the inguinal ligament), thoracic erector spinae spinae (5 cm laterally to T9 spinous process), lumbar erector spinae (3 cm laterally to L3 spinous process), and multifidus (2 cm laterally to L4–L5 spinous processes). The EMG signals were band passed between 20 and 500 Hz, amplified, and sampled at 1.6 kHz.

Electromyographic Ratios. Electromyographic signals were filtered with an adaptive filter algorithm to attenuate ECG contamination[31] and subsequently full-wave rectified. Stability is considered to be most problematic in nearly upright postures.[30] Therefore, data analyses were concentrated on the range around the upright. Data obtained over a range of motion from −5° to 5° in each plane of movement were averaged.

Forces obtained in the ramp contractions were converted to moments using measured distance between the harness and the L4–L5 interspace. Electromyographic data from the beginning of the ramp up to the instant when 17 Nm of moment was produced (the lowest maximum moment attained among all subjects) were averaged. Data from the two repeat trials were averaged. Subsequently, three ratios of EMG amplitudes were calculated: ratio 1 (sum of antagonists over sum of agonists), ratio 2 (lumbar erector spinae [LES] over thoracic erector spinae [TES]), and ratio 3 (internal oblique [IO] over rectus abdominus [RA]). Because of the small moment arms of most muscles in the transverse plane, their functions with respect to twisting efforts is ambiguous, as evidenced by EMG data.[32] In addition, because gravity does not cause moments during the torsion trials, net moments are very low. These factors do not allow unambiguous assignment of EMG signals to either agonistic or antagonistic muscle function. Therefore, ratio 1 was not calculated for torsion trials.

Moment Ratios. An EMG-driven trunk muscle model was used to estimate moments produced by the muscles of interest. The geometry and outline of the model have been described earlier.[33,34] In the current study, 98 vectors crossing the L4–L5 joint were used to represent internal and external obliques and rectus abdominus[35] as well as erector spinae and multifidus muscles.[36] Muscle forces were estimated as the product of maximum muscle stress, 2.5-Hz low pass-filtered EMG amplitudes,[37,38] and correction factors for instantaneous muscle length and contraction velocity.[39,40] Conventionally, the EMG signals are normalized to MVC values, and maximum muscle stress is iteratively adjusted to obtain maximum agreement (least squares) between the time series of muscle moments and net external moments.

In the current model, no MVC normalization was used. Data from the ramp contractions up to 15 Nm of moment produced were used to estimate gains for each muscle group represented by one EMG electrode pair. This gain represents the maximum muscle tension divided by the maximum EMG amplitude. Data obtained at low moment levels only were used because in the conditions to which the model was applied, similarly low moments were produced. Constrained optimization was used to get these estimates, with the cost function being the sum of the squared difference between the net moment components measured and the corresponding muscle moment components predicted by the model. This fit was optimized for all eight ramp contractions at the same time. The constraints were based on the assumption that maximum tension is between 20 and 100 Ncm^{-2}, and that the maximum EMG amplitude would be larger than the maximum found in the (complete) ramp trials and lower than three times this maximum. A third constraint limited the difference between the maximum and minimum of the maximum muscle tension estimates for the different muscles to a factor 2. After the gains had been estimated, the model was applied to the EMG data of the motion trials and ramp contractions to obtain estimates of the moments produced by agonists and antagonists, TES and LES, and RA and IO. From these moments, ratios corresponding to the aforementioned EMG ratios were calculated.

Statistical Analysis. Analysis of variance was applied for motion trials and ramp contractions, and for each ratio separately. Within-subject factors were load (for motion trials only), plane, and direction of movement or attempted movement. Between-subject factors were gender and health status. In all tests, results were considered significant at a P value less than 0.05.

Table 2. Overview of Significant Effects on the EMG Ratios as Revealed by the Analysis of Variance

Condition	Dependent Variable	Factor/Interaction	Test-Statistic		*P*
Motion	ratio 1 (antag/agon)	status	$F_{1,28}$	9.00	0.006
		gender	$F_{1,28}$	5.57	0.025
		plane	$F_{1,28}$	26.95	0.00003
		direction	$F_{1,28}$	10.58	0.003
		load	$F_{1,28}$	8.52	0.007
		plane * status	$F_{1,28}$	8.07	0.008
		gender * direction	$F_{1,28}$	5.66	0.024
	ratio 2 (LES/TES)	status	$F_{1,28}$	8.77	0.006
		plane	$F_{2,56}$	4.16	0.021
	ratio 3 (IO/RA)	plane	$F_{2,56}$	12.42	0.00003
		direction	$F_{1,28}$	4.95	0.034
Ramp	ratio 1 (antag/agon)	plane	$F_{1,28}$	7.49	0.011
		direction	$F_{1,28}$	21.60	0.00007
		plane * direction	$F_{1,28}$	21.59	0.00007
	ratio 2 (LES/TES)	status	$F_{1,28}$	8.43	0.007
		direction	$F_{1,28}$	5.77	0.023
		plane * direction	$F_{1,28}$	6.92	0.014
	ratio 3 (IO/RA)	plane	$F_{1,28}$	22.18	0.00006

antag = antagonistic; agon = agonistic; LES = lumbar erector spinae; TES = thoracic erector spinae; IO = internal oblique; RA = rectus abdominus.

Model Simulations. From two healthy subjects, repeated MVC contractions in several directions were obtained using manual resistance. These were used to obtain normalized EMG amplitudes for motion trials and ramp contractions. Data from the two subjects were averaged to create a data set representative of a healthy subject and fed into a spinal stability model. This model has been described in detail previously.[30] In short, MVC-normalized EMG amplitudes are used to estimate muscle force and stiffness for each of the 90 muscles represented in the model. The stability index quantifies the curvature of the system's potential energy in the vicinity of the static equilibrium. The index is a function of the stiffness in each of the 18 degrees of freedom in the model (6 lumbar intervertebral joints times 3 rotations).

Subsequently, the EMG data were manipulated to simulate an increase in each of the three ratios separately. For ratio 1, antagonistic activity was increased by 20%. For ratios 2 and 3, the activity of TES and RA were decreased by 50%. The activity of the other muscles was increased to keep the average lumbar moment equal.

■ Results

Electromyographic Ratios

The hypotheses formulated on the basis of the first two EMG ratios (sum of antagonists over sum of agonists and lumbar over thoracic erector spinae) were supported by the analysis of variance (Table 2). In the motion trials, the ratio of agonist over antagonist EMG amplitudes was significantly higher in the patient group. The ratio of LES over TES EMG amplitude was higher in both motion and ramp trials. The hypothesis regarding ratio 3 (IO over RA EMG amplitude) was not supported. The main effect of health status (LBP or no LBP) on ratio 3 was not significant ($F_{1,28} = 0.395$; $P = 0.535$), nor were there any significant interactions with health status. Figure 1 provides an overview of the main findings for ratio 1. In the motion trials, the interaction effect of status and plane of movement is readily apparent, with the difference between patients and controls much more pronounced in sagittal plane movements. A tendency toward an effect of status on the ramps can be seen only for flexion ramps. Figure 2 shows that the effect of status on ratio 2 is consistently present across all conditions.

Moment Ratios

After optimization, the model in general predicted the muscle moments reasonably well. Coefficients of determination (R^2) ranged from 0.49 to 0.93 (median, 0.77).

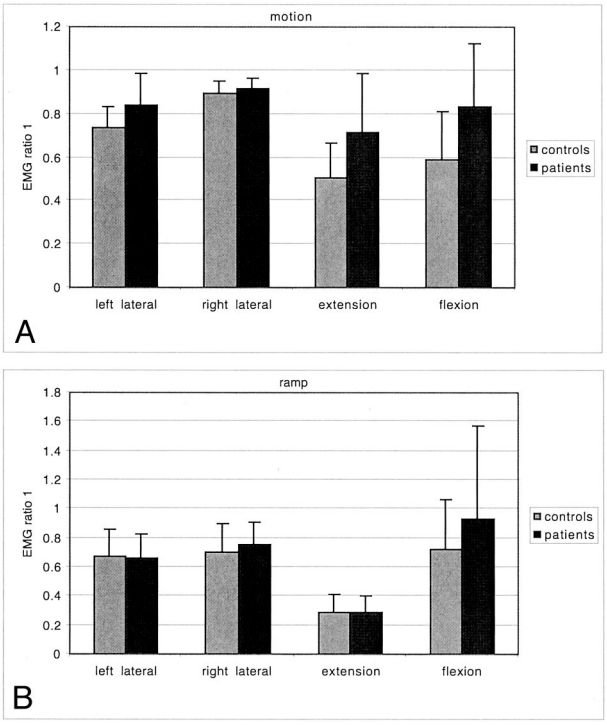

Figure 1. Electromyographic (EMG) ratio 1 (antagonist over agonist EMG) in motion (**A**) and ramp (**B**) trials. Values shown are averaged across loaded and unloaded conditions and across subjects. The error bars indicate one standard deviation of the mean.

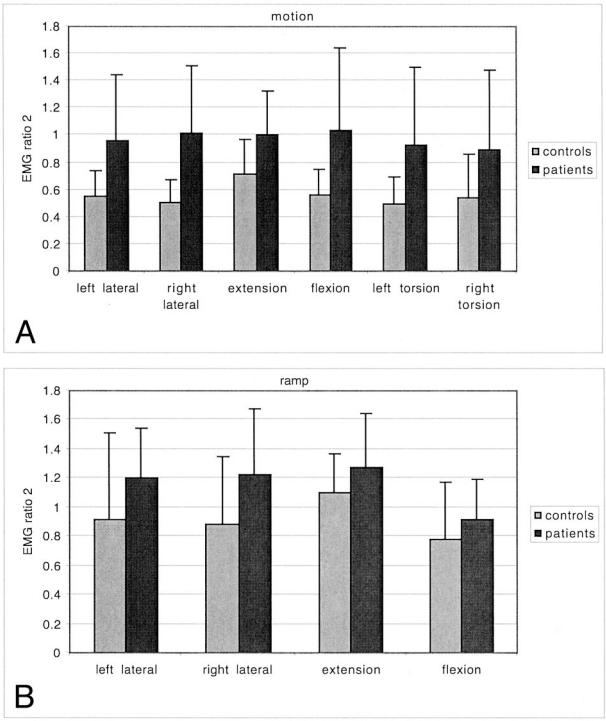

Figure 2. Electromyographic (EMG) ratio 2 (LES over TES EMG) in motion (**A**) and ramp (**B**) trials. Values shown are averaged across loaded and unloaded conditions and across subjects. The error bars indicate one standard deviation of the mean.

This corresponded to averaged absolute errors ranging between 1.8 and 5.9 Nm (median, 3.6 Nm), which was 6.9% of the net moment. The goodness of fit was not significantly different between the patients and the control subjects (difference between medians, 0.005; Mann–Whitney $U = 128$; $P = 1.0$).

The model predicted an increased ratio of antagonist over agonist moments in the motion trials, but not in the ramp trials (Figure 3, Table 3). As shown in Figure 3, the effects of health status on ratio 1 were clearly present only in the sagittal plane motion trials. In the sagittal plane ramp trials, a tendency toward higher antagonistic moments in the patients was present. The moment contribution of the LES relative to that of the TES was significantly higher in the patient group than in the control group in both motion trials and ramp contractions (Figure 4, Table 3).

Simulation Results

Increases in each of the three ratios were predicted to increase stability of the lumbar spine (Figure 5). The effect generally was strongest for the increase in the ratio of LES over TES activity. Simulation of all three increased ratios simultaneously had an additive effect and resulted in the largest enhancement of spine stability. Overall stability was higher in the ramp trials than in the motion trials.

■ Discussion

The current study compared trunk muscle recruitment patterns between patients with LBP and control subjects. Systematic differences found between the two groups appear to reflect a trunk muscle recruitment strategy that serves to increase spinal stability in the patients. During the experiments, the study participants experienced no pain. Therefore, direct effects of pain can be excluded.

Recruitment patterns were first analyzed *via* ratios of EMG amplitudes of different muscle pairs. A comparison of raw EMG amplitudes may be confounded by, for example, increased subcutaneous fat related to a less active lifestyle in patients because EMG amplitudes recorded at the skin will decrease with an increase in the thickness of the fat layer as a result of tissue filtering. Electromyographic ratios, however, are not likely to be confounded by such differences between groups. Selective increases in subcutaneous fat over the abdominal and lumbar areas would decrease rather than increase the ratios studied. The ratio of LES over TES activity is used as a measure of moment contribution of intersegmental in relation to multisegmental muscles on the assumption that the lumbar EMG electrodes mainly record activity from segmentally inserting muscles whereas the thoracic electrodes mainly represent activity of muscles inserting on the pelvis and thorax only. This appears to accord with anatomic data presented by Macintosh and Bogduk.[41] Likewise, ratio 3 assumes representation of segmentally inserting (internal oblique) muscle activity by one electrode pair and representation of activity resulting from a muscle inserting on the pelvis and thorax only (rectus abdominus) by the other electrode pair. Given their locations, crosstalk (*i.e.*, contamination of the recorded

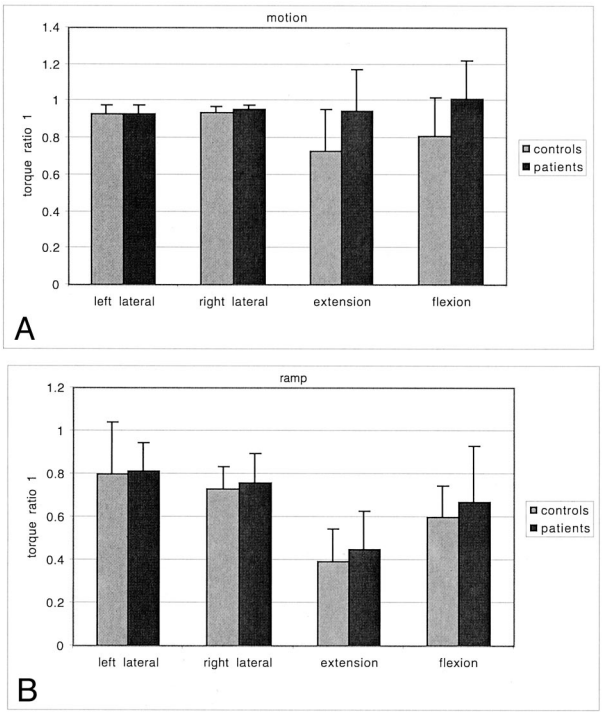

Figure 3. Moment ratio 1 (antagonist over agonist moment) in motion (**A**) and ramp (**B**) trials. Values shown are averaged across loaded and unloaded conditions and across subjects. The error bars indicate one standard deviation of the mean.

Table 3. Overview of Significant Effects on the Moment Ratios as Revealed by the Analysis of Variance

Condition	Dependent Variable	Factor/Interaction	Test-Statistic		P
Motion	ratio 1 (antag/agon)	status	$F_{1,28}$	5.24	0.030
		direction	$F_{1,28}$	6.37	0.018
	ratio 2 (LES/TES)	status	$F_{1,28}$	5.01	0.033
	ratio 3 (IO/RA)	plane	$F_{1,28}$	5.55	0.006
Ramp	ratio 1 (antag/agon)	plane	$F_{1,28}$	56.85	0.00000
		direction	$F_{1,28}$	7.90	0.009
		plane * direction	$F_{1,28}$	25.35	0.00003
	ratio 2 (LES/TES)	status	$F_{1,28}$	5.02	0.033
		plane * direction	$F_{1,28}$	5.75	0.023
	ratio 3 (IO/RA)	plane	$F_{1,28}$	23.43	0.00004

antag = antagonistic; agon = agonistic; LES = lumbar erector spinae; TES = thoracic erector spinae; IO = internal oblique; RA = rectus abdominus.

signal with signals from another muscle) from these muscles is unlikely. However, the information picked up by the IO electrode pair is likely to be contaminated by activity of the external oblique muscle (EO). Although anatomic descriptions support the classification of the IO as a segmentally inserting muscle,[42] this is more ambiguous for the EO muscle. In addition, crosstalk, in this case from the transverse abdominus muscle, may occur. This muscle, however, inserts segmentally as well.

The EMG-assisted model used to estimate the moment ratios resulted in fairly good predictions of muscle moments. In some individuals, less accurate predictions were obtained. However, this occurred both in the control and patient groups, and thus would not have systematically affected the comparisons. Average performance of comparable models using MVC-normalized EMG amplitudes were similar.[43,44] Given the low moment levels used, these results are satisfactory. A recently published model that also uses nonnormalized EMG as input[45,46] relies on estimates of MVC based on anthropometrical data not available in the current group of subjects. This model showed higher R^2 values and better correspondence with net moments than the current model. However, it was tested at much higher moment levels (lifting loads) and in symmetric conditions only. Goodness of fit between the total muscle moment and the net moment was improved in the current model, as shown when the muscle moments were fitted to the

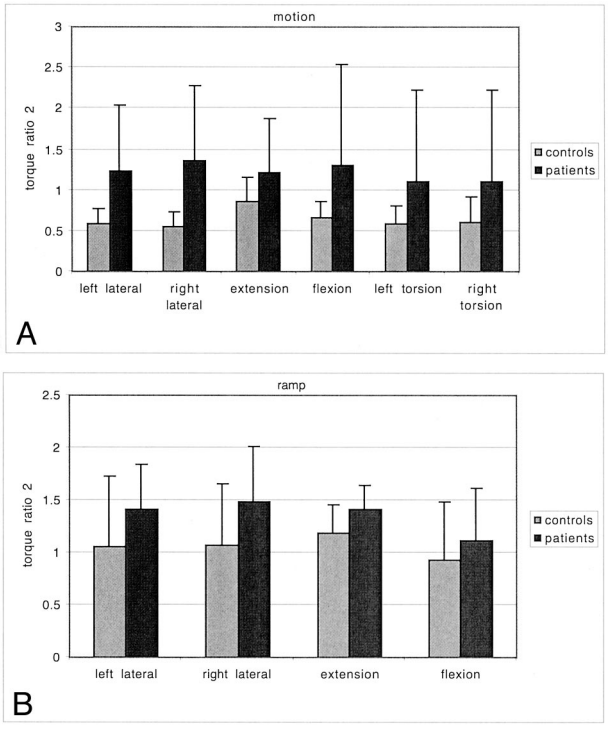

Figure 4. Moment ratio 2 (LES over TES moment) in motion (**A**) and ramp (**B**) trials. Values shown are averaged across loaded and unloaded conditions and across subjects. The error bars indicate one standard deviation of the mean.

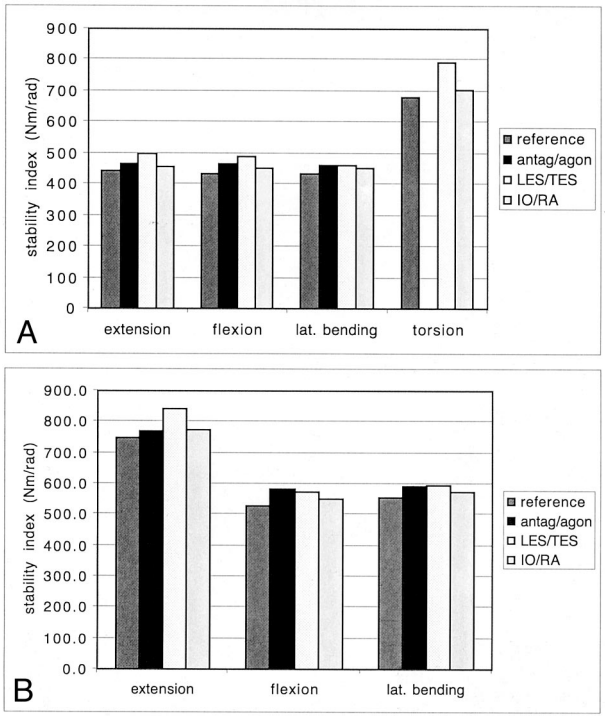

Figure 5. Spinal stability (stability index) calculated for the motion (**A**) and ramp (**B**) trials. Stability that resulted from the generic "healthy" electromyographic (EMG) pattern was used as a reference. Subsequent simulations were performed with altered EMG recruitment patterns by increasing each of the three ratios as hypothesized to occur in patients with low back pain. Results were averaged over the unloaded and loaded conditions.

complete net moment series of the ramp trials instead of up to 15 Nm only. However, this procedure probably will limit accuracy of predictions at specific (in the current case low) moment levels. The use of a model with nonnormalized EMG signals may tend to obscure differences in recruitments if these are present during calibration trials also. When, for example, a subject displays a high-level antagonistic activity during the calibration trial, fitting the model may result in low gain values, especially for the antagonist muscles. This problem can be avoided only by using a balanced set of calibration trials in which moments are produced in opposite directions.

The most systematic effect found was the larger moment contribution of the LES relative to the TES contribution in the patients than in the controls. The simulations performed support the assumption that this effect is functional in terms of increasing stability or compensating for a loss of stability otherwise. An alternative explanation might be that an increased activity of lumbar muscles is required to compensate for selective wasting of these muscles in patients with low back pain.[47] However, whereas this would affect EMG ratios, moment ratios should not be affected. Differences in lumbar curvature may have occurred that could perhaps underlie the increase in LES recruitment. This possibility cannot be excluded. However, the angles between the trunk and pelvis were controlled, so major changes in curvature can be ruled out. Moreover, this would not affect the conclusion that the muscle recruitment pattern observed in the patients does enhance spinal stability. In apparent contradiction to the current results on ratio 2, Larivière *et al*[48] found a significantly higher TES EMG amplitude in patients with LBP than in healthy control subjects during several loaded and unloaded movements. However, this increase was found only in the left side TES, and similarly raised amplitudes were found in the left LES and latissimus dorsi muscle, although these did not reach significance.

Effects on ratio 1 expressing the level of antagonistic activity were less consistent, and no effects on ratio 3 (IO over RA activity) were found. The inconsistent effects on ratio 1 can be explained by several factors. First, the effect was not significant during ramp contractions. This may be explained by the fact that in these trials the cable providing resistance enhances stability in very much the same way as antagonistic muscle force would. This explanation is supported by the simulations, which indicate a higher stability in the ramps trials than in the motion trials. Second, the effect appeared to be more pronounced in sagittal plane motions and ramps than in frontal plane trials. This may be explained by the fact that cocontraction levels during lateral bending were already high in the control subjects, possibly related to the presence of a forward bending moment caused by gravity.

No significant effects of health status on ratio 3 were found. This ratio was proposed by O'Sullivan *et al,*[49] and actually shown to be reduced in patients with LBP during specific exercises. The simulations predicted an increase of IO over RA activity to be effective in increasing stability, although much less so than an increase in LES over TES activity. Furthermore, the effect of IO activity on stability probably is manifested mainly in the frontal plane.[42] Because passive trunk stiffness is higher in the frontal plane than in the sagittal plane,[50] frontal plane stability may not be a limiting factor in the patients.

Besides the positive effect of a change in trunk muscle recruitment patterns, some negative consequences may occur. Increased activity, related to increased cocontraction, could cause pain in the muscles themselves, contributing to a vicious circle of pain–spasm–pain.[2] In addition, increased cocontraction would increase the forces acting on the spine.[51] Furthermore, selective derecruitment of the TES may limit functional abilities of patients. Finally, it is possible that changes in recruitment of trunk musculature remain present after their functional significance has disappeared, because injured structures have recovered. In chronic low back pain, aspects of pain behavior in many cases appear to remain although the physiologic cause may no longer be present.[52,53] Nevertheless, caution should be exercised when patients with low back pain are rehabilitated, with sole purpose of restoring a normal muscle recruitment pattern. The "abnormalities" may represent compensation mechanisms to stabilize the spine. Future studies should attempt to discriminate between causal/contributing and functionally adaptive neuromuscular factors associated with low back pain.

■ Key Points

- Patients with low back pain and control subjects performed trunk motions and isometric contractions.
- Ratios of EMG amplitudes and estimated moment contributions of antagonist over agonist muscles, and of segmentally inserting muscles over muscles inserting on the thorax and pelvis only were calculated.
- The ratios of antagonist over agonist and of lumbar over thoracic erector spinae EMG amplitude as well as the corresponding ratios of estimated moment contributions were greater in patients than in control subjects.
- Simulation showed that the recruitment pattern found in the patients enhanced spinal stability.

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